

# Peptic Esophagitis

## Its Surgical Significance

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■ *Esophagitis caused by the reflux of acid gastric juices through an incompetent sphincter at the junction of the esophagus with the stomach is now recognized as a common cause of upper abdominal and thoracic symptoms frequently simulating heart, stomach or gallbladder disease. While these symptoms are occasionally mild and transient, under certain circumstances permanent, irreversible and seriously disabling changes may occur in the lower esophagus. The usual cause is a sliding hiatal hernia. Although mild symptoms can often be relieved by simple means, advanced disease may require a major surgical procedure to relieve scar tissue stricture of the esophagus which may eventuate and permanently disturb the swallowing mechanism.*

*Hiatal hernia causing displacement of the cardioesophageal junction, the most common cause of esophagitis, can be corrected either by transabdominal or transthoracic surgical procedures directed toward reduction of the hernia. Fixation of the stomach to the abdominal wall in a position of downward traction has been used as a simple and successful means of correcting the hernia.*

SINCE peptic esophagitis was first described by Winkelstein<sup>9</sup> in 1935 as a new clinical entity, there has been a growing interest in this problem. Only within the past few years, however, has the medical profession recognized the full significance of esophageal irritation as a cause of upper abdominal and thoracic complaints. It is now well known that regurgitation esophagitis represents the most common nonspecific inflammatory process in this

organ. While symptoms may be relatively mild and fleeting when associated with transient cardioesophageal incompetence, under certain conditions reflux may lead to permanent, irreversible and seriously disabling changes in the lower esophagus. Experimentally, the esophagus, which is lined with squamous epithelium, has been shown to be less resistant to peptic digestion than any other portion of the gastrointestinal tract.

Under normal conditions the esophagus is protected from the regurgitation of acid gastric contents by a sphincter mechanism at the esophageal

Presented before the Section on General Surgery at the 93rd Annual Session of the California Medical Association, Los Angeles, March 22-25, 1964.

agogastric junction. Unfortunately, we are so wanting in knowledge regarding the operation of this mechanism, either in health or disease, that there is no unanimity of opinion about the basic anatomic and physiologic features of normal gastroesophageal continence.

For an understanding of the clinical problems associated with reflux of gastric juice into the esophagus, consideration must be given to the normal state and the changes that lead to incontinence. Of especial interest because of its structure and function is the portion extending from the upper limit of the ampulla to the esophagogastric junction, a segment making up approximately 20 per cent of the length of the organ. The level of the hiatus does not seem to help in defining the position of the underlying esophageal anatomic structure, nor does the diaphragm play any known coordinate part in the apparent specific functions of the lower esophageal structures.

The well-known controversy as to the presence or absence of an anatomically distinct sphincter at the cardia is less important to the clinician than the well-documented fact that there obviously is a physiological sphincter mechanism in this area which prevents the reflux of gastric contents and which apparently utilizes certain unspecialized circular muscle segments to cause segmental constriction readily demonstrable by esophagoscopy and radiological examinations. These observations are readily acceptable to most investigators.

In addition to these intrinsic factors, there appear to be other mechanisms involved, most prominent of which is the angle of entrance of the esophagus into the stomach, which under normal conditions forms a flap-like type of valve thought by some investigators to play a major role in the prevention of regurgitation.

Although the true nature of an anatomical sphincter has not as yet been proven, it is now well known that disturbances in the physiological sphincter mechanism, allowing recurrent or prolonged regurgitation of gastric contents into the esophagus, lead to inflammation and ulceration and ultimately to fibrosis and stenosis. Fibrosis will not only narrow the lumen of the esophagus, but eventually will cause longitudinal shortening. Here, again, opinions differ as to cause and effect. Many observers believe that congenital shortening of the esophagus leads to disturbances in the sphincter mechanism with resultant esophagitis and stricture, while others consider the shortening the result of fibrosis secondary to inflammation. Suffice it to say, for purposes of the present communication, that shortening associated with inflammation, fibrosis and stricture does occur in infants as well

as in adults, and the therapeutic problem entailed is extremely difficult.

The primary pathophysiologic feature in reflux esophagitis is an incompetent cardiac sphincter. Because of sphincter incontinence, the regurgitation of gastric chyme occurs. The development of esophagitis and its complications is a secondary phenomenon. The inflammatory process usually is limited to the lower portion of the esophagus and particularly to that portion immediately above the cardiac orifice. In the early stages, granular erythema with edema manifests itself. Then, as the disease progresses, ulceration develops. The ulcers tend to be superficial and serpiginous. However, in the more advanced stages, large ulcers may eventuate which occasionally penetrate, causing localized fixation. A thin, yellow surface slough with underlying granulation tissue forms over the affected area. As such lesions bleed easily, chronic anemia often results. Progressive submucous fibrosis eventually leads to stenosis and stricture formation which may extend over an extensive area, resulting in decided shortening of the esophagus. As shortening occurs, the stomach is pulled upward into the thoracic cavity. Microscopically, there is epithelial necrosis and hyperplasia with pronounced degrees of polymorphonuclear infiltration and varying degrees of fibrosis, depending upon the chronicity and severity of the process.

Peptic esophagitis in the milder forms may be transient and is often associated with physiological processes leading to sudden or prolonged increases in intra-abdominal pressure. The most noteworthy of such processes are obesity and pregnancy. The acid regurgitation creating heartburn in the late stages of pregnancy is a common phenomenon and probably is related to a temporary incompetence of the cardiac sphincter brought about by the displacement of the abdominal organs and undue pressure on the stomach. Although distressing, this situation is relieved by delivery and does not lead to permanent changes in the esophagus. Milder degrees of peptic esophagitis are often encountered in association with gallbladder dysfunction and peptic disease of the duodenum. In these circumstances, pylorospasm with gastric retention and regurgitation accounts for the symptoms. Relief depends upon control of the basic disease of the gallbladder or duodenum.

Most commonly, however, this entity is observed in association with the sliding type of esophageal hiatal hernia, with surgical excision or destruction of the cardiac sphincter or with persistent vomiting. Occasionally it occurs following the prolonged use of indwelling nasogastric tubes.

In our experience, persistent vomiting and the use of nasogastric suction are not common causes

of peptic esophagitis. However, when the disease does occur from such causes the process is apt to be fulminating in nature, to develop rapidly and to be very severe. Ulceration with bleeding and the early formation of strictures are not uncommon.

The development of esophagitis in association with hiatal hernia and following operations upon the cardia is more insidious and slowly progressive. Any operation which disturbs the esophagogastric sphincter mechanism may eventually lead to prolonged and repeated regurgitation, with the eventual development of peptic esophagitis and its sequelae. Most of the cardioplastic procedures carried out in the past for the treatment of achalasia, at first thought to have been successful because of the relief of cardiospasm and obstruction, eventually led to reflux esophagitis and its complications. Such also has been the case too frequently following excision of the cardia with restoration of gastrointestinal continuity by esophagogastrotomy.

By far the most common defect demonstrable in association with esophagitis is that of a hiatal hernia of sliding type which allows elevation and displacement of the cardio-esophageal junction. The esophagus is shortened and does not extend to the level of the diaphragm. While there is still a great diversion of opinion regarding the causes of the incontinence at the cardia in hiatal hernia, my own studies seem to support the opinion of other investigators that the angle of entrance of the esophagus into the stomach is disturbed, thus obliterating one of the most important factors in the prevention of regurgitation. It is well known that not all hiatal hernias cause symptoms; in fact, some of the largest and most easily demonstrable do not. However, it is important to recognize that there is a great variation in the anatomical relationship between the defective hiatus and the esophagus, resulting in involvement of different structures and in the development of hernias of different types. It would appear from my own observations as well as those of other investigators that it is only when the esophagogastric junction has been displaced, altering the angle of entrance of the shortened esophagus into the stomach, that regurgitation and esophagitis ensue. In my experience the symptoms associated with para-esophageal hiatal hernia, in which the angle of entrance of the esophagus into the stomach has been preserved, are due to pressure and are not the symptoms associated with esophagitis. In contradistinction, in the sliding type of hernia with a disturbed esophagogastric angle, the symptoms are almost exclusively attributable to esophagitis. In the presence of ulcer diathesis, with or without a demonstrable peptic ulcer, reflux greatly aggravates the symptoms.

The symptoms of peptic esophagitis are variable, depending of course upon the stage of the disease and the etiological factors. In the milder forms, periodic and transient degrees of heartburn may be the only presenting symptom. As the disease progresses, esophageal spasm may cause regurgitation and vomiting with substernal discomfort on swallowing. Progressive anemia may be an associated factor. Bleeding, while frequent, is not usually massive or severe. However, occasionally massive upper gastrointestinal hemorrhage has been described, particularly in the presence of acute ulceration. When a sliding hernia is present, the symptoms may appear mainly at night with the patient recumbent and may be relieved by his assuming a sitting or upright position. During the day, forward bending may initiate symptoms.

When cardiospasm occurs, or particularly when progressive fibrosis and stenosis develop, dysphagia with nausea and vomiting, mainly of solid foods, becomes prominent and makes differentiation from carcinoma difficult.

The diagnosis depends upon roentgenographic and esophagosopic findings. Physical signs are absent except for varying degrees of inanition as the disease progresses.

Inflammation, spasm, ulceration and fibrosis may all be recognized by a trained endoscopist. However, since all of these findings may be present in combined inflammatory and malignant disease, esophageal biopsy becomes an essential part of examination.

X-ray studies may be relatively diagnostic and the findings will depend upon the stage of the disease. Roentgenographic changes are usually absent in the early stages of hyperemia and mild inflammation. However, even early in the course of the disease there may be a fine irregularity without a clear delineation of the normal mucosal pattern. Esophageal spasm may be evident in the more advanced cases. Varying degrees of esophageal stenosis occur typically just above the cardia. The transition from the proximal esophagus, which may be dilated, to the strictured area usually is gradual and symmetrical. Esophageal ulcer may be demonstrable as well as other associated pathologic change. The presence of a hiatal hernia of sliding type is easily overlooked; when such a condition is suspected, special technique should be instituted to demonstrate it.

From the therapeutic point of view, peptic esophagitis presents an equal challenge to internist and surgeon. Prophylaxis is extremely important in light of the irreversibility of the late changes that may occur. It is important to recognize the etiological factors concerned, the better to anticipate the progress of the disease process which may follow

and to try to prevent it rather than treat the irreversible condition. Medical management is in most respects, similar to that instituted to control the acid secretions in peptic disease of the stomach and duodenum. In addition, attempt should be made to minimize reflux by urging good posture upon the patient, reduction of body weight and avoidance of compressing garments. Bending at the waist should be avoided, as should sleeping completely prone. When these measures fail or when the etiologic factor is an anatomically correctable defect, early surgical correction with the restoration of normal anatomical relationships may prevent more serious changes and avoid the major surgical procedures necessary for the correction of esophageal stricture.

Bouginage may be a useful adjunct to treatment, particularly in the presence of cardiospasm; however, it should be recognized that dilation of fibrous strictures, while perhaps permitting food to pass more readily, may at the same time aggravate the basic condition by permitting regurgitated gastric juice to reach higher levels in the esophagus.

The surgeon's role in the treatment of this disease is an important one. As he is usually not called upon until the disease has reached an advanced stage, he should be interested in its prevention. He is the main offender as regards injudicious or prolonged use of nasogastric suction. In anticipation of this problem, temporary gastrostomy rather than nasogastric suction, when prolonged suction is anticipated, should be recognized as a safe and acceptable procedure. The surgeon should appreciate the significance of gastroesophageal reflux and its sequelae, as well as the fact that any surgical procedure which destroys the esophagogastric mechanism will eventually lead to reflux and its complications. He, therefore, should avoid operations upon the esophagogastric junction unless it is already so disturbed that it no longer may act as a sphincter or is so diseased that it may interfere with the life of the patient.

When it becomes essential that the cardioesophageal junction be revised, it must be appreciated that, in the absence of a sphincter, regurgitation will occur. Further degrees of esophagitis can be prevented only by selecting a surgical procedure which will either prevent postoperative gastroesophageal reflux or will render the patient achlorhydric and still maintain gastrointestinal continuity.

Surgical repair of a sliding esophageal hiatal hernia is indicated in most cases of reflux esophagitis when these conditions coexist and is, by all means, the most successful therapeutic procedure applicable under these circumstances. Unfortunately, however, surgical repair of a hiatal hernia is of no avail if delayed until after stricture and

shortening have occurred. Indeed, in these circumstances it is usually impossible, because of the shortening secondary to fibrosis, to restore the normal position of the cardia at the level of the hiatus.

Confronted with such circumstances, the surgeon must resort to more radical operative procedures, most of which are hazardous and associated with a high rate of morbidity and mortality. The area of stricture must be resected and gastrointestinal continuity must be reestablished. However, when the cardia is resected the sphincter mechanism is destroyed, and unless it is restored by some means, regurgitation will eventually lead again to esophagitis and its sequelae. Although many ingenious methods have been proposed to reestablish a sphincter in this area, none have been completely successful.

In the presence of advanced stricture formation, the simplest means of repair is esophageal resection with reestablishment of gastrointestinal continuity by esophagogastrostomy. Unfortunately, although the stricture is removed by this procedure, gastroesophageal reflux persists and recurrent esophagitis frequently occurs above the site of anastomosis. Pyloroplasty, as an adjunct to this procedure, tends to reduce the amount of regurgitation by allowing free egress of gastric contents into the duodenum.

Resection of the esophageal stricture, accompanied by resection of the acid-bearing proximal stomach, with esophagogastric anastomosis between the antrum and proximal esophagus, will prevent acid regurgitation but is frequently complicated by the late development of intractable type of anemia.

Vagotomy and antral resection after resection of the esophageal stricture, with re-anastomosis of the proximal stomach to the esophagus and the distal stomach to the duodenum, has been reported as a successful means of treatment. Acid secretion, in these circumstances, is controlled by the antrectomy and vagotomy; however, gastroesophageal reflux still occurs, and it remains to be seen whether or not the esophageal mucosa will tolerate the regurgitation of alkaline gastric contents.

In recent years a widely used method of reestablishment of continuity between the esophagus and stomach has been the interposition of a segment of jejunum, as advocated by Merendino.<sup>7</sup> He showed that such a small bowel segment actually functions as a sphincter, preventing reflux of stomach contents into the esophagus. When anatomically feasible, this operation is acceptable, but it is particularly hazardous because of the precarious blood supply to the transplanted jejunal segment.

Because of technical problems associated with an interposed jejunal segment, many surgeons more

recently have been utilizing colon rather than small bowel as the interposed segment. Time alone will determine the success or failure of this procedure. I have had particular success with this method in the management of caustic strictures of the esophagus, but am not prepared, from personal experience, to recommend it for the management of advanced cases of peptic esophagitis.

Fortunately, in the great majority of cases of peptic esophagitis the disease has developed in association with a hiatal hernia of sliding type and the symptoms usually are relieved by adequate repair of the hernia. Although the results of repair of esophageal hiatal hernia by either a transpleural or transperitoneal approach are generally satisfactory, by no means does either method consistently bring about complete relief of symptoms. Bradshaw recently reported persistence of symptoms in 16.5 per cent of cases after repair, exclusive of cases in which there was recurrence. Brintnall<sup>4</sup> reported treatment failure in 42 per cent of cases.

Because of experiences such as these, a number of different methods of surgical repair have been suggested. Until recently, most methods of repair were fashioned after the transthoracic approach of Allison or the transabdominal route of Harrington.<sup>6</sup> Numerous modifications of these techniques have been devised, most of which include fixation of the cardio-esophageal juncture at its normal level in relation to the diaphragm and approximation of the crura of the diaphragm in front of or behind or lateral to the esophagus. Most recently, operations have been proposed which, in addition to correcting the anatomical defect, include methods devised to reduce gastric acidity and promote rapid emptying of the stomach.<sup>2</sup>

It is noteworthy that both Nissen<sup>8</sup> of Germany and Boerema<sup>3</sup> of Holland have reported excellent results from very simple surgical repairs directed only toward fixation of the cardio-esophageal juncture at its normal level in relation to the diaphragm by suture of the stomach to the under-surface of the diaphragm. By this means, they attempt to restore the angle of entrance of the esophagus into the stomach. In their hands this operation prevents reflux and effectively relieves symptoms.

In 1960, Colonel H. H. Ziperman<sup>10</sup> and I became interested in this simple approach after it was noted at operation that a sliding hernia was easily reduced by caudad traction on the lesser curvature of the stomach. As long as this traction was maintained, the peritoneum, which originally constituted the hernia sac, seemed to bridge the defect in the hiatus, preventing further herniation. When the traction was released the hernia recurred. We had long been impressed by the futility of attempting

to prevent herniation by simple closure of the crura of the diaphragm about the esophagus, particularly in view of the necessity of leaving a defect sufficiently large to protect the esophagus against constriction. Indeed, in the past we have often found the crura so attenuated that it was impossible to approximate them effectually about the esophagus. However, in these circumstances a repair directed only toward reestablishment of the cardio-esophageal angle proved effective. It also occurred to us that if indeed the angle was the important factor as regards the sphincter action, and this angle could be reestablished and maintained by fixed traction on the lesser curvature of the stomach. Gravity, when the patient is in the upright position, should be an aid in maintaining traction, as opposed to our usual inability to keep organs in the proper position against the force of gravity.

For these reasons it seemed worthwhile to attempt repair by simple fixation of the lesser curvature of the stomach to the anterior abdominal wall in a position entailing sufficient tension that one could no longer force one's finger into the hiatal defect. This was accomplished by forcefully pulling the stomach on its lesser curvature downward and forward until the hernia defect was no longer palpable and then fixing the stomach in this position to the anterior abdominal wall with a series of silk sutures. This procedure was first attempted in a patient who had a hiatal hernia incidental to other abdominal disease. Following operation the hernia was no longer demonstrable. Since then we have used this simple method in many patients with hiatal hernia, particularly when the hernia was found in conjunction with other intra-abdominal disease and we were reluctant to subject the patient to repair of a more extensive type. To date, the results have been most gratifying. The preliminary results and follow-up on a group of patients treated by this simple maneuver have been reported elsewhere.<sup>10</sup>

For a good-risk patient in whom the diagnosis is certain and there is no other intra-abdominal disease, the most reasonable procedure would seem to be transthoracic repair from above the diaphragm with particular emphasis on fixation of the cardio-esophageal junction at the level of the diaphragm. Too frequently, however, this ideal situation does not exist and laparotomy is essential to a reasonable diagnosis. In these circumstances, particularly in obese or elderly patients or when other intra-abdominal procedures must be undertaken, it may be anticipated that an attempt to fix the gastroesophageal junction and stomach from below the diaphragm, to say nothing of the approximation of the crura, may be difficult and possibly hazardous. Perhaps many of the surgical failures

reported may be attributable to poor repairs under these conditions. We have chosen, in these patients, to utilize the simple pull-down procedure described.

Sometimes for a patient who has a healed or active peptic ulcer in addition to a hiatal hernia we choose vagotomy, pyloroplasty and gastrostomy high on the lesser curvature with fixation of the stomach at the site of the surgical opening to the anterior abdominal wall in a position of caudad traction. This effectively reduces the hernia and maintains the reduction. The few patients who have been treated in this manner have been particularly pleased with the results and we have been impressed with the simplicity of the operation and the uneventful convalescence.

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